technical aspects of stent deployment in the cephalic arch could be shared with others. We are keenly aware of the need to preserve "venous capital" but have not found stenting of the cephalic arch to be a factor in its diminution. This is borne out by our life-table analysis for a 1 year functional patency of 100%.[1]

Stent placement in the cephalic arch, as elsewhere, is an attempt to preserve patency of an existing access. Our study showed that covered stents demonstrated significantly better patency than bare stents.[1] The rate of restenosis requiring dilatation in the stent graft group was half of that in the bare stent group. This was a significant improvement but, as Dr Turmel-Rodrigues states, redilatations are still required despite covered stent placement. In fact, in the constellation of conserving venous capital, dilatations and redilatations are required to keep accesses open and to increase dialysis time for any particular access, stented or not.[3]

Our study clearly showed that the use of bare stents for cephalic arch restenosis does not prevent rapid in-stent restenosis. Stent grafts performed significantly better and are the preferred solution for this lesion with improved patency and decrease in re-intervention rates (Fig). It is logical to extrapolate from this study that bare stents are not an appropriate solution for any venous lesion in arteriovenous accesses that require stents and that covered stents will do better. Moreover, we would encourage the use of stent grafts that are completely covered and flexible. Needless to say, we think it incorrect to apply reported anecdotal findings concerning bare stent occlusions in central veins to the deployment of stent grafts in cephalic arch restenosis. We do not see any reason to prohibit their use in this circumstance.

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Regarding "Impact of calcification and intraluminal thrombus on the computed wall stresses of abdominal aortic aneurysm"

We read with great interest the article by Li et al, concerning the effect of intraluminal thrombus on the values of peak wall stress, in 3D reconstructed individualized abdominal aortic aneurysm (AAA) models.[1] After having used the method of Finite Element Analysis, the authors concluded that the relative amount on thrombus reduces the maximum stress in AAA in a rather linear way ($r = 0.863$, $P < .001$). This is one of the largest series (20 patients) with a rather interesting finding, adding to the existing information about the biomechanical influence of thrombus on maximum stress and consequently on the risk of rupture.[2]

Although the amount of thrombus is sufficiently addressed, we believe that an additional point needs to be considered, namely the concurrent influence of the geometric modifications inside the lumen that the existence of thrombus induces, when compared with reconstructed AAA models without thrombus. These shifts can be represented by the alterations in the mean curvature, torsion, tortuosity in the lumen centerlines in each case.[3]

In our laboratory, we also used the Finite Element Analysis method and confirmed the good level of correlation between the reduction in maximum stress and the relative amount of thrombus in a series of 19 patients (Spearman's non parametric $r = 0.5$, $P = .03$). However, when we used a partial correlation analysis (non parametric Spearman test), enabling us to control for the difference in the geometry parameters that have shown to have an influence on maximum stress magnitude,[4] we failed to show any statistical significance for the relationship between stress reduction and the relative amount of thrombus (Spearman's $\rho = 0.413$, $P = .112$). This may imply that the protective role of thrombus is not only a matter of amount, but could be influenced by the geometry of its distribution. This observation could offer a new insight into the limitations of the protective role of thrombus.

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Reply

We thank Dr Georgakarakos et al for the interest they have shown in our study.[1] They confirmed our finding in their study that a good correlation existed between maximum wall stress and the relative amount of thrombus. However, by employing partial correlation analysis and controlling the geometric variations, they reported that the difference between the above was not statistically significant. This might imply that peak wall stress was affected by the geometric variations rather than the amount of thrombus alone.

We would like to impress on the point that although this may be the case, the geometric variation is itself dependent on the volume and location of the thrombus. The concentricity or eccentricity of thrombus by changing the centerline curvature can alter the peak wall stress. The location and volume of thrombus may also affect the pattern of aneurysm expansion.[2,3] There is an analogy between the role of thrombus in altering the aneurysm wall stress and the role of calcification in atheromatous plaques. Conventionally, the amount of calcification was used as an indicator of plaque vulnerability but our group has shown that location is another key determinant of plaque vulnerability.[4] Similarly, we hypothesize

that in addition to the volume of intraluminal thrombus, its location and impact on the geometry of the arterial lumen can affect the wall stress and ultimately result in variable pattern of aneurysm growth. A prospective longitudinal is therefore required to assess the changes in thrombus volume, resultant change in lumen curvature, and pattern of aneurysm growth/expansion, before we can say that tortuosity of lumen centerline is the key determinant of increasing the aneurysm wall stress.

Another important area worth exploring is the material behavior of intraluminal thrombus. It is most likely a non-homogeneous material with a complex property. A large ex vivo experiment is needed for future study in this area to improve our understanding of aneurysm material properties and failure strength. This can help in creating more realistic computational models, which could be used as a clinical adjunct in the future for effective decision making in aneurysm repair.

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Regarding "Risk-adjusted 30-day outcomes of carotid stenting and endarterectomy: Results from the SVS Vascular Registry"

We have read the study by Sidawy et al[1] about 30-day outcomes of carotid artery stenting (CAS) compared with endarterectomy (CEA) with great interest. Unfortunately, we are forced to conclude that major flaws in the study prevent drawing meaningful conclusions from this observational cohort study. Although cohort studies can sometimes be powerful tools in assessing treatment effectiveness,[2] the authors' study fails to meet minimal standards for such studies; most importantly, (1) complete and unbiased follow-up of study end points, and (2) rigorous control for confounders.

The validity of the conclusions drawn from a study such as theirs, with only 44% follow-up, is extremely limited. Those lost to follow-up are likely to be less adherent to concomitant drug therapy and are often more likely to have had complications; or in contrast, sometimes those who are doing extremely well may waive follow-up visits.[3-5] In general, 80% follow-up in longitudinal studies is considered a minimum, and >90% follow-up is generally feasible in short follow-up studies like that of Sidawy et al.[1] This study's poor follow-up is made worse by the different follow-up rates between groups, by the reliance on self-report, and by the presence of systematic differences between CEA and CAS follow-up, because Centers for Medicare and Medicaid Services rules require in-hospital results for CAS for recertification, whereas there is no such mandate for CEA.

Furthermore, controlling for all potential confounders is always important in cohort studies, but particularly in those in which the choice of intervention is heavily influenced by patient factors (ie, "selection").[2] Those who are more frail or who have higher surgical risk are generally much more likely to have a minimally invasive procedure (CAS) instead of a surgery (CEA), and biases due to such patient and provider selection are notoriously difficult to adjust for in cohort studies, usually requiring special methods such as instrumental variable analyses.[2]

The results of Sidawy et al are in conflict with several randomized controlled trials comparing these two interventions and reporting equivalence of the two interventions,[6-9] even in the long-term.[6] Therefore, we suggest that the short-term differences found in this observational cohort study with poor follow-up and likely inadequate control for confounding do not provide useful evidence on this important clinical topic.

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Reply

The authors appreciate the comments from Drs Meier and Hayward indicating potential flaws in our article, the first being lack of complete and unbiased follow-up and the second being inability for rigorous control for confounders. These weaknesses were already identified as issues inherent to a study based on